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Abstracts

Symposium 3: Evolution and diversity of pattern

Program/Abstract # 17

Towards understanding evolutionary diversification in leaf form

Miltos Tsiantis

Department of Plant Sciences, University of Oxford, UK

A key problem in biology is to understand how variation in organismal form is generated. The basis for phenotypic diversification of reproductively isolated species has been difficult to study because of the paucity of experimental systems where the developmental genetic changes underlying such variation can be accurately identified. To investigate this problem we study the genetic mechanisms underlying variation in form of the predominant photosynthetic organ of plants, the leaf. Leaf form can be classified as simple, where the leaf blade is entire as in the model organism *Arabidopsis thaliana*, or dissected where the blade is divided into distinct units called leaflets. Mechanisms that determine specification of dissected versus entire leaf shape and regulate the number, position and timing of leaflet production are poorly understood. To obtain an in-depth and unbiased understanding of these mechanisms we established *Cardamine hirsuta* – dissected leaf relative of *A.thaliana* – as a powerful experimental system where both forward and genetics, and stable genetic transformation can be deployed for studying diversification of leaf morphology. Here I will discuss how comparisons between *A. thaliana* and *C. hirsuta* have illuminated our understanding of processes underlying evolution of plant form.

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Program/Abstract # 18

Widespread gene expression divergence between organisms with near-identical embryonic development

Itai Yanai, Craig P. Hunter

Department of Molecular and Cellular Biology, Harvard University, Cambridge, MA, USA

Evolution of developmental processes can be associated with specific changes in gene expression. However, differences in gene expression between species do not necessarily indicate adaptive changes in form and function; changes in gene expression may evolve instead by non-adaptive processes. Here we use organism-specific microarrays to compare the embryonic transcriptomes of two morphologically near-identical nematodes, *Caenorhabditis elegans* and *C. briggsae*, whose genomic sequence divergence is on par with that within mammals. We find that of the most highly conserved genes expressed in both embryos over a third exhibit divergent temporal gene expression patterns. Genes with essential embryonic functions exhibit significantly more gene expression conservation, while genes unrelated to development are enriched for expression divergences. The same pattern is found in comparing two strains of *C.*

elegans, indicating similar evolutionary forces on gene expression at the population level. We find that overall gene expression divergence is correlated with genomic disturbances, in terms of local promoter composition changes and global gene order rearrangements. However, essential genes do not conform to this pattern suggesting that they are robust to genomic changes. Finally, we find that complex expression patterns involving multiple induction times evolve at a considerably faster rate than relatively simple expression patterns as would be expected by a modular model of gene expression. Our results indicate a major role for non-adaptive processes on the control of gene expression of even highly constrained developmental processes.

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Program/Abstract # 19

Non-equivalent in vivo function for mouse and zebrafish Hoxa3 genes using gene targeting in mice

Nancy R. Manley, Lizhen Chen

Department of Genetics, University of Georgia, Athens, GA, USA

Hox genes encode a family of transcriptional factors that have conserved functions in patterning the antero-posterior axis during embryogenesis in all bilaterian animals. Duplication of Hox clusters and their recruitment in evolutionary novelty has been an active subject of study. Swaps of the 60 amino acid homeodomain from different mouse paralogous Hox groups revealed that different paralogous groups have evolved distinctly. On the other hand, Hox proteins from the same paralogous group have been shown functionally equivalent. Expression of vertebrate Hox ortholog proteins in *Drosophila* have been reported capable to rescue Hox mutants in *Drosophila*, suggesting that Hox proteins are functionally conserved cross the phyla. We used gene targeting to express the zebrafish Hoxa3a gene in the place of the mouse Hoxa3 gene. The zebrafish Hoxa3a protein could complement the Hoxa3 null mutant phenotype in the some tissues, such as thyroid and trachea, but formed novel skeletal structures, and was unable to complement the development of thymus and parathyroid. Molecular analysis showed that zfHoxa3a protein was unable to upregulate Pax1 expression in the endoderm, a molecular target linked to athymia and aparathyroidism in Hoxa3 mutants. Thus the zebrafish Hoxa3a protein is functionally distinct to the mouse Hoxa3 protein despite of the highly conserved homeodomain. These data suggest that Hoxa3 protein has evolved significantly since the last common ancestor of mouse and zebrafish, and suggests the existence of neofunctionalization of Hox proteins is a key aspect in the evolution of novelty during vertebrate evolution.

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